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THE MEDICAL JOURNAL OF AUSTRALIA.

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ON HEART ACTION AND MURMURS.[1]

By **J. Macdonald Gill, M.D. (London),**
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My intention to-night is to discuss the origin of murmurs from a purely theoretical point of view. No apology is needed for that, as the practical and the theoretical are, or should be, one. I am convinced that the proper understanding and treatment of heart disease is greatly hindered by a wrong idea of the way in which murmurs in the heart arise. Nor does it avail one to endeavour to ignore them by saying that in most cases they have no significance. In the past, no doubt, exaggerated importanee was attached to them. But the fact remains that they exist in most cases of heart trouble and should be studied and explained.

I shall describe and discuss, first, the murmurs heard in acute rheumatism in childhood, particularly first attacks. The commonest is, of course, the apical systolic. Sometimes, when faint, it disappears after rest in bed, but generally it returns after exercise. Varying greatly in loudness, its essential characters are similar in most cases. It may, if loud, be audible over a large area of the thorax. Speaking very generally, a loud murmur means a badly damaged heart. The next most common murmur is the diastolic. This is most common at the apex and is always combined with a systolic. It is a *diminuendo* murmur, seldom loud, often faint and never conducted widely over the thoracic wall; it is therefore strictly limited in its area of audibility. It may occupy the whole diastolic period, so that the second sound is not audible. In other cases, the second sound is quite clear and is followed after an interval by this murmur, which is itself separated from the following first sound by a short pause. It is, therefore, properly described as a "mid-diastolic" murmur. This murmur, in first attacks of rheumatism, may disappear very quickly. In other cases, more advanced, when the heart is a good deal hypertrophied, one hears at the apex during diastole a rumbling, echoing sound, difficult to describe. In one case there was audible at the apex a loud systolic murmur, but away from the apex, towards the lower end of the sternum, a continuous tingling sound, not a murmur. These two murmurs, the apical systolic and diastolic, are the only murmurs at all common in early attacks of rheumatism. Aortic murmurs, systolic or diastolic, are rarely heard in children under twelve years of age. Probably one would be right in saying that they are never heard in first attacks of rheumatism. They more usually show themselves after several attacks and in the later years of childhood. Murmurs are sometimes more audible over the veins of the neck than over the heart. They are usually systolic in rhythm and are very often present. Murmurs in the pulmonary area are not so frequent nor so loud as those at the apex.

[1] Read at a Meeting of the New South Wales Branch of the British Medical Association on September 24, 1920.

How are these murmurs produced? It is first of all necessary to consider the pathology of rheumatism. In a child dead from rheumatic heart disease after a short illness, which generally means pericarditis, there is, as a rule, very little valvular disease; in fact, the valves may be perfectly healthy. The vegetations in recent cases are small and situated along or near the margin of the valves. They are incapable of either obstructing the flow of blood from the auricle to the ventricle or of preventing the two flaps of the mitral valve from coming together on contraction of the ventricle. This, of course, has long been known and led to the idea of "muscle failure," as it has been called. It was thus supposed that there was a yielding of the muscular tissue around the mitral orifice, which readily gave way to pressure, owing to the injury caused by the rheumatic poison. It is often supposed that there is a general dilatation of the left ventricle, but that is an error. The apex beat may be in the normal position and the cardiac dulness may be normal. Moreover, the murmur may remain unaltered for years, without any signs of heart failure. There is clearly no general dilatation of the ventricle. One is led to suppose, then, that the muscular tissue around the mitral orifice yields to the increased intraventricular pressure produced by the contraction of the main mass of the ventricle. To make this theory probable, one must suppose that there is a special band of muscular tissue forming a sphincter around the mitral orifice. Such a band of muscle has never been described. Nor does it seem reasonable to suppose that the rheumatic poison would damage this band and leave the rest of the ventricular muscle free. Still, of course, such an occurrence is possible. The diastolic murmur has still to be explained. Some writers appear to think that such a murmur must mean mitral stenosis. But it is often heard in first attacks of rheumatism, when mitral stenosis is never present. Graham Steell, in his book on "Heart Disease," describes two cases in detail, showing diastolic murmurs in which at the *post mortem* examination no mitral stenosis was found. He frankly admits that he could not explain the occurrence of a diastolic murmur in such cases. Any explanation, to be satisfactory, must account for both the systolic and the diastolic murmurs. The muscle failure idea, as explaining the systolic murmur, must be regarded as quite inadequate. Any satisfactory theory should also explain the murmurs audible in the veins of the neck.

We will now take the murmurs heard in anæmia. Murmurs are usually audible in chlorosis, pernicious anæmia and the severer forms of secondary anæmia, following hæmorrhage. They are not usually present in leuchæmia. In all these cases, the same murmurs are heard and are no doubt due to similar causes. The "venous hum" is best heard over the right internal jugular or bulb and frequently over the left innominate vein, as it rises up behind the episternal notch. It is often heard over the left internal jugular. It is often accompanied by a thrill. It is in its most

complete form a continuous humming or roaring sound. If not audible in the recumbent position, it may become readily so by getting the patient to sit up in bed or to stand up. In cases where recovery takes place, it will gradually disappear. In mild cases, instead of being continuous, it may be only systolic. The pulmonary area is the next most common seat of murmurs in anæmia; in this, it is always systolic and is often accompanied by a systolic pulsation, due to the infundibulum of the right ventricle. Systolic murmurs are also often heard at the apex and more rarely in the aortic area. All these murmurs disappear on recovery.

I shall now take the presystolic murmur into consideration. This murmur stands by itself and needs special consideration. In the past, it has been the subject of an enormous amount of discussion. The question in dispute has been, not its mode of production (the point we are discussing to-night), but its position in the cardiac cycle, *i.e.*, whether it is of ventricular or auricular origin. I think that everybody is now agreed that it is of auricular origin. But it is a mistake to suppose that it is always due to mitral stenosis, or dependent upon contraction of the left auricle. It is not infrequently heard in auricular fibrillation, when there is no proper contraction of the auricle. In cases of aortic regurgitation, the "Austin-Flint" murmur may resemble the presystolic murmur in every way, so much so as to lead experienced physicians to diagnose aortic regurgitation, when none has been present. In the same way, it may be heard not infrequently in cases of adherent pericardium. It is also described as having occurred in chronic Bright's disease. Osler mentions a case in which it was heard where the heart was displaced by an enormously enlarged spleen. It is obvious that none of the explanations ordinarily in vogue can explain all these cases.

Now I shall refer to the "to and fro" aortic murmur. Many years ago, Finlayson described two cases in which this murmur was heard, but the aortic valves were perfectly normal. In aortic aneurysm a murmur is never present, unless the aortic orifice is involved.

Now, what is the explanation of all these murmurs? Recent writers on heart disease seldom discuss the exact manner in which murmurs are produced. They assume, apparently, that all murmurs are of mechanical origin. The fluid vein theory is generally supposed to explain them all. The "venous hum" of anæmia is admittedly difficult to explain in this way. It is supposed that the internal jugular vein is narrowed when passing through the deep cervical fascia and dilated both above and below this opening. This is obviously highly improbable, as the murmur is best heard when the patient is in the erect posture, the vein being then collapsed and empty. Neither does it explain why the murmur is continuous, the flow of blood in the vein not being continuous, as shown by graphic records. This is the only explanation I have seen suggested for this murmur. It is clear that it is in some way dependent upon the anæmic state of the blood, as it disappears when recovery sets in. My own idea is that the vibrations which produce the murmur are of muscular origin, the muscular tissue producing them being situated at the junction of the superior *vena cava* and the right auricle. This muscular tissue contracts rhythmically at a perfectly regular rate, usually eight per second. These rhythmical contractions are normally very feeble, but in anæmia they become palpable in the neck and produce a continuous humming sound. One must remember the distance travelled is very short, 2.5 to 5 cm. at most, in a perfectly straight line. I think that the vibrations are transmitted to the neck partly by the wall of the vein and partly by the blood itself. I think it is probable that the lessened viscosity of the blood in anæmia is a factor.

The pulmonary systolic murmur is now to be considered. The explanations given in the text-books are utterly inadequate. One is that a fluid vein is here produced by dilatation of the pulmonary artery, owing to the anæmia. The late Dr. Foxwell, of Birmingham, suggested that a kink in the wall of the artery was produced by the dilated right ventricle pushing up the pulmonary orifice, the bifurcation being a fixed point. There is no doubt but that the infundibulum of the right ventricle is apt to be dilated in chlorosis, as a pulsating area, systolic in time, is often seen at the inner end of the second left intercostal space. I think that the explanation is simple. The dilated infundibulum transmits the vibrations I have already described to the chest wall, just as the jugular vein does to the neck. A certain degree of tension of the muscle is necessary, as the vibrations would be lost when the muscular tissue was flaccid; no doubt, too, the blood stream conveys the sound.

A similar explanation applies to the common systolic apical murmur. During systole, the ventricles impinging against the chest wall would transmit the vibrations communicated to them by the auricle. The diastolic portion of a "to and fro" murmur is simply caused by the apex withdrawing somewhat from the thoracic wall after the contraction of the ventricles is over.

I think that the mid-diastolic murmur has probably a different origin. The vibrations producing this murmur may possibly come from the left auricle, being conveyed by the blood stream from the left auricle to the apex. I think that similar vibrations to those arising from the junction of the superior *vena cava* and the right auricle may arise from the pulmonary veins at their juncture with the left auricle.

The presystolic murmur must now be considered. I think that it is produced in a similar way to the mid-diastolic, the additional factor being hypertrophy of the left auricle. The blood stream conveys the vibrations to the apex; as soon as the contraction of the ventricle begins the murmur stops. The left auricle acts as a resonator. The increasing tension of the left auricle in mitral stenosis during diastole would facilitate the production of a murmur. The final accentuation of the presystolic murmur has been plausibly attributed to the contraction of the auricle. This cannot be so, as a typical murmur of this kind may be heard in auricular fibrillation when the auricle is inactive.

Electro-cardiograms favour this view, as the *P* wave often shows one or two notches in mitral stenosis, as well as being more prominent.

Aortic murmurs must be considered next. I think that the dilated aorta is the chief factor. In some cases the vibrations are probably transmitted direct to the chest wall from the auricle. In one case, for instance, of aortic regurgitation in a man, a thrill could be felt to the right of the sternum, over quite a large area, far beyond the situation of the aorta, as much diastolic as systolic. It was no doubt a continuous vibration and could only have come from the right auricle.

It is now necessary to discuss the origin of these vibrations in greater detail. That they are of muscular origin is obvious. That this muscular tissue is of a primitive character is also obvious, rhythmical activity being characteristic of such tissue. We have in the heart two kinds of muscular tissue, the ordinary striated muscle of the auricle and ventricles and the smooth muscular tissue of the auriculo-ventricular bundle. It is also probable that such tissue exists at the junction of the superior and inferior *vena cava* with the right auricle and of the pulmonary veins with the left auricle. The anatomical details have not yet been fully worked out, except in the case of the "*a-v*" bundle. We have, therefore, in the heart two kinds of muscular tissue, as well as two kinds of muscular movement. It is reasonable to suppose that the more elementary tissue retains its activity, when the more developed is exhausted or paralysed. Hence, in auricular fibrillation, the fibrillary movement is due to the persistence of the rhythmical activity of the primitive muscular tissue, after the auricles are paralysed; a similar condition may exist in the ventricles. This seems to me to be the simplest way of explaining auricular or ventricular fibrillation. The "*a-v*" bundle is admitted by all to possess some of the characteristics of muscular tissue, *viz.*, conduction, excitability and obviously tone. It is reasonable to suppose that it possesses the others, *viz.*, rhythmicity and contractility. The last function is obviously the most important of muscular tissue. Corresponding to the double musculature of the heart we have a double innervation, vagus and sympathetic. The sino-auricular node is probably connected with the right vagus and the "*a-v*" node with the left. The close relation of the sino-auricular node with the superior *vena cava* and of the "*a-v*" node with the coronary sinus points to that. The superior *vena cava* is the homologue of the right horn of the *sinus venosus* and the coronary sinus of the left horn. It is impossible to accept the view that the heart beat originates in the sino-auricular node, as the inferior *vena cava* and the coronary sinus, as well as the pulmonary veins, must be taken into account. The sympathetic is the motor nerve of the striated muscle and the vagus the motor nerve of the smooth muscular tissue of the heart.

The physiological basis of these remarks is of a most striking nature. Gaskell's book on the "Involuntary Nervous System," published in 1916, shortly after his lamented death, furnishes us with the evidence we need on the physiological side. I now quote from Gaskell;

> Fano, in 1900 found that, when records were taken of the contractions of the auricle and ventricle of the water tortoise (*Emys europœa*), a clamp being placed at the auriculo-ventricular groove, the base line of the auricular tracing was not straight, but slowly undulating, the rhythm of the waves being often remarkably regular. The auricle of this tortoise shows the presence of two rhythmical activities going on simultaneously, the one a tonic rhythm, having the appearance of "Traube-Hering" curves, and the other the ordinary heart beats superimposed on this tonic rhythm. Fano at first looked upon this phenomenon as indicating the presence of two kinds of muscle in the auricle, the one the ordinary quickly contracting cardiac muscle, the other a more slowly contracting kind, which, in consequence of the clamp in the auriculo-ventricular groove, was set into rhythmic action. He drew attention to the fact that atropine removed these rhythmic tonic contractions, while the heart beats continued, but muscarine removed the heart beats, while the tonic rhythm continued.

These observations were confirmed and extended by Bottazzi:

> Rosenzweig, working with Engelmann, found that the clamp at the "*a-v*" groove was not necessary for the production of this tonic rhythm, but that bloodlessness and a dying condition of the heart were sufficient to bring about the phenomenon. Rosenzweig further examined the heart of *Emys europœa* histologically and found that close against the endothelium was a layer of muscular tissue in which no striation could be seen. He ascribed the tonic rhythmical contractions to this layer. Bottazzi found the two kinds of contraction behaved differently to stimulation of the cardiac nerves; stimulation of the augmentors inhibits these tonic contractions, so that the auricular contractions all start from the same base line, while, of course, the beats themselves are increased in strength. Conversely, stimulation of the vagus will make the tonic rhythm appear, if it is not present, and increase the strength of the waves if they are weak, while, as is well known, it diminishes the strength of the auricular beats. . . Bottazzi's statements agree in a remarkable way with Fano's observation upon the action of atropine and muscarine upon these two kinds of rhythm, for atropine is essentially a paralyser of the activity of muscular structures, to which the vagus cells send motor fibres, while it leaves alone or even increases the activity of muscular structures, whose motor fibres belong to the sympathetic cells; and muscarine paralyses the latter but not the former. Since Rosenzweig's paper, Bottazzi has re-examined the question. . . He confirms his previous observations on the respective actions of the two cardiac nerves and also shows that adrenaline produces the same effect upon the two rhythms as stimulation of the sympathetic. Oinuma has confirmed Bottazzi's observations on the cardiac nerves and I have seen the same action of adrenaline as he describes upon the auricle of *Crysomis picta*, an American species allied to *Emys*. Bottazzi also, in conjunction with Ganfin, has examined histologically the auricular tissue of *Emys* and has confirmed Rosenzweig's discovery of unstriped muscle fibres in the auricle. They form a strong layer next to the endothelium and continue into the *sinus venosus* and the beginnings of the great veins. The striated muscle fibres also continue into such a vein as the *vena hepatica*, always lying external to the layer of unstriped muscle. In agreement with its histology, the *sinus venosus* exhibits a well-marked tonic rhythm, which is influenced by the cardiac nerves in the same way as that in the auricle.
>
> These observations definitely prove the existence in the heart of a system of unstriped musculature, in addition to the striated cardiac muscles, which is especially well developed in the water tortoises and extends from the auricles into the beginnings of the great veins. This unstriped muscle resembles the enteral unstriped muscles, the bronchial muscles and those of the gall bladder in its behaviour to poisons like atropine

and muscarine and in its innervation; for all these muscles are supplied with motor fibres from cells connected with the vagus outflow and with inhibitory fibres from cells connected with the thoracico-lumbar (*i.e.*, sympathetic) outflow. On the other hand, the striated cardiac muscle resembles the enteral sphincter muscles, in that their motor nerve cells are connected with the thoracico-lumbar (or sympathetic) outflow and their inhibitory cells with the vagus outflow. In fact, exactly the same kind of reciprocal innervation exists in the heart as in the intestine.

Gaskell assumes, of course, that this unstriped muscle had disappeared in the higher vertebrates, as it had never been found in them. But I think we are justified in assuming that such a muscular tissue would not altogether disappear. The evidence I have given from the clinical side justifies us in thinking that it must still be present in the human heart, although further research from the anatomical side is necessary.

EPITHELIOMA OF THE LIP.

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(From the Walter and Eliza Hall Institute, Melbourne.)

Foreword.

In this paper the nature of the primary growth in 62 cases of precancerous, early and intermediate stages of epithelioma of the lip was investigated. The cases were selected from the practice of the Melbourne Hospital during the last four years. In any large hospital epithelioma of the lip occurs frequently and ample opportunities exist for the study of the disease in all its stages.

Sex Incidence.

Sixty-one cases occurred in males, one in a female.

Site.

Sixty of the lesions were situated on the lower lip, one at the angle of the mouth and one on the upper lip (in a female).

The Cancerous Site.

The usual site for cancer of the lip is at the transition line from skin to mucous membrane or close to this line. Here the hairs disappear and the epithelium becomes more transparent. The outer cells are still cornified, but this cornification disappears soon after the transition line is passed. The free surface of the epithelium of the mucous membrane is smooth. Its deep surface is indented with long papillæ. Upon the mucous surface of the lip the epithelium rests on a connective tissue layer, the *tunica propria*, which corresponds to the corium on the skin surface.

On both the skin and mucous surfaces the epithelial layer consists of a thick stratified epithelium. The deepest portion, consisting of basal cells, rests upon a definite basement membrane. The basal cells are columnar in shape and have elongated, well-staining nuclei. The cell outline is indistinct. There is comparatively little cytoplasm surrounding the nucleus. Above the basal layer is a layer of cuboidal cells, which are larger than the basal cells and have a more distinct outline. The nuclei are rounded and do not stain so deeply as the basal nuclei. No intercellular bridges are seen in either this or the basal celled layer. Above the cuboidal celled layer, which is only about two cells thick, is the squamous celled layer. There is no sharp transition between these two layers, the cells of one merging into those of the other. In the squamous layer the cells are large, polygonal in shape and arranged generally in a mosiac pattern. Intercellular bridges are present. The nuclei are rounded; the surrounding cytoplasm is abundant and clear. Towards the surface the cells become flattened and the intercellular bridges less marked. In the skin the uppermost cells are pigmented with fine granules (*stratum granulosum*). In the mucous membrane the flattening is not marked and there are no pigment granules. Above the epithelium on the skin surface is a clear layer (*stratum lucidum*) and above this again a cornified flaky layer (*stratum corneum*). On the mucous surface there is no *stratum lucidum* or *corneum*, these being represented only by a slight cornification of the surface cells.

Neoplasms Arising from the Epithelium of the Lip.

In 1904 Krompecher (1) pointed out that an epithelial neoplasm may consist of cells resembling either basal, cuboidal or squamous cells. The basal celled growths are the least malignant and the squamous celled growths the most malignant. The basal celled growths embrace the rodent ulcer group. Growths arising in the cuboidal celled layer are not common. Bloodgood (2) investigated these cases and reported 40 in all situations. Three occurred in the lip. He remarks that it is often very difficult to distinguish them from basal celled growths and thinks that mixed cases must occur. Among the cases diagnosed as rodent ulcer of the lip in the Melbourne Hospital during the past four years, I was unable to distinguish any cuboidal celled growths. They were all true basal celled growths. Malignant growths consisting of squamous cells are true squamous celled carcinomata or epitheliomata and it is with this class of growth that this paper deals.

The Precancerous Condition.

Whether or not an epithelioma can arise in normal epithelium is still *sub judice*. It is certain, however, that in a large number of cases there is a pre-existing abnormality which in the case of the lip may take the form of leukoplakia, syphilis or chronic hypertrophic irritation due to pipe smoking. At the present stage of our knowledge a precancerous condition cannot be defined by the criterion that unless the cause is removed it will go on to malignancy, for beyond the fact that chronic irritation is a factor, the cause is not known. In the case of epithelioma a precancerous condition may be defined as one which presents histologically a state of chronic inflammation with epithelial hypertrophy at some stage between normal epithelium and definite early epithelioma. In some cases this may go on to epithelioma, in others it does not. I have produced similar lesions in the dog and guinea-pig by means of X-rays. These have cleared up.

The clinical importance of a precancerous condition is the fact that it may lead to cancer. In this series it was found that in the usual type of precancerous lip there was a proliferation of the squamous cells, with projection of the deep surface of the epithelium into the underlying stratum. The basal cells proliferated to form an apparently normal basal layer. The cuboidal celled layer was distinguishable. The basement membrane was intact. Projections of epithelium, either fine or massive in type, were pushed down. Each projection retained its normal relationship of basement membrane, basal cells, cuboidal cells and squamous cells.

In another less common type the basal cells proliferated to a greater extent than was necessary to form a layer of normal thickness. The basal celled layer was hypertrophied either uniformly or in places. In some cases solid buds of basal cells were seen at the ends of the epithelial projections.

Figure I. shows two epithelial projections in a precancerous lip. In one there is a more or less normal layer of basal cells, with cuboidal cells and squamous cells above them. Intercellular bridges and the mosiac-like arrangement of the squamous cells are seen. In the other projection there is a marked proliferation of basal cells, which form a bud-like mass at the tip of the projection. What the factors are that determine the amount of basal cell proliferation one cannot say.

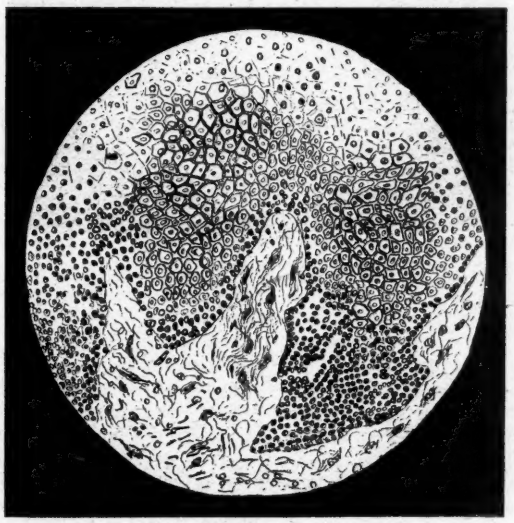

FIGURE I..
Precancerous condition in epithelioma of lip. An uncommon type, with marked basal cell proliferation.

Six cases were classed as precancerous; in four of these the basal celled layer appeared to be normal, in one somewhat thickened and in one markedly hypertrophied, the buds of basal cells above referred to being marked. This is the case illustrated in Figure I..

Stage One.

Very Early Epithelioma.—The criterion of malignancy is the breaking through of the basement membrane by the epithelial cells. The transition is not abrupt and there occur many doubtful cases. The basement membrane normally separates the basal cell layer from the cells of the corium or *tunica propria* and appears as a fibrous layer to which the basal cells are attached. The projections or down-growths of epithelium give rise to a number of solid down-growing alveoli, varying in size, but roughly either fine or massive in type. These have broken through the basement membrane and are infiltrating the corium or *tunica propria*. The basal cells, if traced down these early alveoli in some cases, will be seen to form a continuous line; in other cases they are missing in places. No whorls are seen as a rule in early cases, but in the massive form there may be a tendency to early whorl formation. The rôle of the basal celled layer is important. Basal celled tumours, *e.g.*, rodent ulcer, benign cystic epithelioma, sebaceous adenoma, etc., are not highly malignant. In an early epithelioma the basal celled layer is to a certain extent a protective layer. This barrier may disappear early or late. Hazen (3) has pointed out that even in an advanced stage, where the down-growths have divided up into numerous alveoli which have no attachment to the epithelium above them, these alveoli may yet have an intact layer of basal cells. Specimens considered later in this series confirm this (see Figure III.).

In the specimen shown in Figure II. the basement membrane has been broken through and the basal celled layer is missing in places. An alveolus is seen with intact basal cells except in one place, where squamous cells are in direct contact with the cells of the corium. The case is an early epithelioma of the type in which the basal cells disappear early.

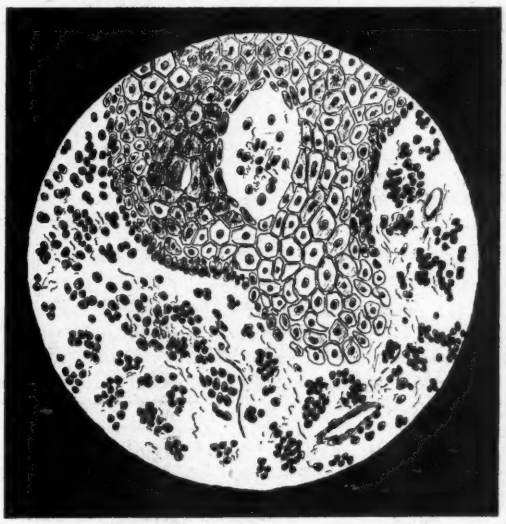

FIGURE II..
Very early epithelioma of the lip, showing early disappearance of basal cells.

There were four very early epitheliomata in the series. In three the basal celled layer was intact. In one (illustrated in Figure II.) the squamous cells were in direct contact in places with cells of the corium.

Stage Two.

In the next stage the early down-growths have spread and pushed out numerous solid alveoli. This stage is not sharply differentiated from the last stage. The down-growing alveoli are continuous with the surface epithelium; that is to say, they have not broken away, as they do later. The basal celled layer, if traced down, is usually found to be missing in places. In some cases it is intact, however. As a rule the basal celled layer keeps pace with the growth through the precancerous stage and through the first stage; it begins to disappear in what I have called the second stage. There is usually some whorl formation. A varying amount of tissue reaction is seen at this and in the early stage. The cells usually seen are small round cells, fixed tissue cells and a few cells resembling mast cells. The cellular reaction is roughly proportional in amount to the rate of growth of the tumour. Four cases were examined at this stage. In three the basal celled layer had disappeared to a varying extent. In one it was intact.

Stage Three.

In the next stage the alveoli which have grown down, begin to break away from their parent down-growths. As the alveolus elongates a constriction appears in some part of it and an island of growing cells is nipped off. This may occur in alveoli which still possess basal cells and the island then appears as a core of squamous cells surrounded by basal cells (as in Figure III.). In other cases the squamous cells have outgrown the basal cells and islands of squamous cells are cut off. In some cases the portion cut off may be partly covered with basal cells. Cell whorls and epithelial pearls become more apparent at this stage. These appearances are simply groups of cells, arranged concentrically and showing an affinity for acid dyes due to degenerative changes. As the degeneration progresses the nuclei disappear and layers of hyaline material arranged like the coats of an onion are seen. This occurs solely in squamous cells and the cells have the appearance of being under great pressure.

In most cases the islands which were cut off, showed no basal cells. There were 30 cases examined at this stage, eight early and 22 late.

Stage Four.

In the next stage the alveoli which are cut off, proliferate widely. The proliferating alveoli in turn subdivide and an extensive infiltration follows. By this time in the majority of cases the basal cells have been outgrown and the alveoli consist of squamous cells only. There is no sharp division between this stage and the last one. It is mainly a question of degree. The microscopic appearances are very similar. In most cases the alveoli were rounded or oval in shape. Numerous whorls and pearls were present. The reaction was usually of the round celled type, though in some cases the fibrous tissue predominated and the growth resembled somewhat a scirrhous carcinoma. The intercellular bridges to a large extent tend to disappear and in parts the cell outlines become indistinct, the nucleus pale and the cell vacuolated.

In other cases the alveoli which were cut off, spread in the form of thin processes, which varied in length and often branched and subdivided.

There is a third type which is uncommon. The basal cells persist. The growth then presents the appearance of a number of more or less rounded alveoli. There is a basal layer; cells resembling cuboidal cells are seen and the central portion consists of a mass of squamous cells with cell whorls and pearls. In the case in which this was most marked, the specimen had been removed after the growth had been present for three years. The patient stated that the growth refused to heal, but was not increasing in size; no glands were palpable. Clinically the course of the tumour resembled that of a basal celled growth; the squamous cells were encapsulated everywhere in an intact layer of basal cells. This case is illustrated in Figure III..

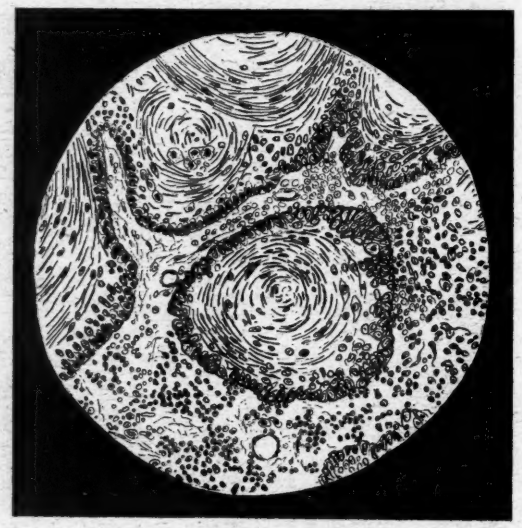

FIGURE III..
Epithelioma of lip, showing late persistence of basal cells.

Conclusions.

The formation of an epithelioma of the lip can be traced through various stages from a precancerous condition to an advanced stage. These stages do not correspond to the age of the growth, for the rate of growth varies greatly. In one of the cases the total history only extended over five weeks and the case could almost be classed as an advanced one.

The causative agent, whatever it may be, stimulates the growth of epithelial cells. Before degenerative changes set in no alteration in the appearance of these cells can be seen. The basal celled layer plays a varying part. In some cases it disappears early and in others is maintained throughout. The reason for this I cannot say. The proliferating epithelium invades the underlying tissues in the form of solid alveoli. These alveoli become detached from the epithelium and form islands which proliferate and subdivide. A varying tissue response has in the meanwhile occurred.

References.

(1) Krompecher—"*Der Basalzellenkrebs*," Jena, 1903, quoted by Hazen in "Skin Cancer."

(2) Bloodgood—*Amer. Jour. Med. Sc.*, 1914, CXLII., 76.
(3) Hazen—"Skin Cancer," St. Louis, 1916.

Reports of Cases.

BRIEF CLINICAL NOTES ON THREE CASES OF INTESTINAL OBSTRUCTION.

By **Archie Aspinall, M.B., Ch.M. (Syd.),**
Honorary Assistant Surgeon and Surgical Tutor, Sydney Hospital.

I submit the reports of the following cases of intestinal obstruction. Intestinal obstruction is not an uncommon malady. Early diagnosis is of vital importance, but at times is extremely difficult. The best treatment to be adopted in the individual case may be the subject of anxiety to the surgeon and can only be decided at the time of operation, after taking everything into consideration.

I am indebted to Dr. E. S. Stokes and Dr. E. Smith for the notes from which the abstracts have been taken. The patients were operated on in the Sydney Hospital.

Case I.—J.I., aged 49, labourer. The patient came to the hospital at 12.45 p.m. on March 16, 1920. As he walked to the Casualty Room he appeared to be in great pain, was "doubled up" every few yards as he walked, and at such times yelled vociferously. On arrival at the Casualty Room he behaved in such an extraordinary way that the Casualty Surgeon could not make a diagnosis and was inclined to think that the patient was not seriously ill, so he referred him to the Out-patients Physician instead of admitting him to Hospital. Whilst at the Out-patients Department he had dry retching and at intervals screamed out. His temperature was subnormal and pulse-rate 50 per minute. The abdomen moved freely and there was no distension. He was tender on palpation, especially in the left lower quadrant. No tumour was felt. He described the pain as starting in the region of the left iliac fossa, radiating to the epigastrium and then back to the point of onset. There was an easily reducible inguinal hernia on the left side. The urine was scanty, but contained no abnormal constituents. The patient stated that he had suffered from attacks of diarrhœa recently, but had gone to bed feeling quite well at night, to be awakened by an attack of vomiting and purging at 1 a.m. and this had continued at intervals since. His previous health had been good.

The patient was admitted to a surgical ward late in the afternoon, when his temperature was 36.1° C. and pulse-rate 100. He was very restless and when questioned, threw himself about the bed and screamed out. The abdomen was distended; the distended bowel gave the abdomen a "ladder pattern." There was no dulness on percussion, but generalized tenderness. Two enemata had been given without result.

At 6.30 p.m., on seeing the patient, I decided to open the abdomen at once. Ether was administered by the open method and the abdomen opened by a median incision below the umbilicus (a rectal tube and catheter were passed without result as soon as the patient was anæsthetized). Fluid escaped from the peritoneal cavity and black, distended bowel presented at once. Volvulus of the small bowel was suspected and a considerable length of distended bowel was pulled out through the wound. It was then found that there was a tight band of omentum fixed to the brim of the pelvis and about 240 cm. of the ileum had passed through and the mesentery was twisted, completely shutting off the blood supply to the affected bowel. The band was easily cut through. After a short time had elapsed and warm pads had been applied to the bowel, there was no improvement in the appearance of the bowel, no pulsation in the vessels. After consideration I decided to excise the bowel and after closing the cut ends, made a lateral anastomosis. The patient was on the operating table for one hour; four hours later the pulse was 126 per minute and the patient warm but restless. His bowels moved later and, although his pulse was feeble, he seemed to have a chance of recovery; but at 10.50 p.m. next day he suddenly died.

Commentary.

(1) The diagnosis in the early stages was difficult; ptomaine poisoning and calculus anuria were suggested. The delay was fatal.

(2) The agonizing pain was apparently due to torsion of the mesentery.

(3) Prior to operation the distended bowel gave the abdomen a "ladder pattern," the rungs being represented by distended bowel.

(4) Vomiting was an early symptom; distension was a late symptom.

(5) With the initial shock the temperature was subnormal and the pulse slow. Later the pulse-rate increased, whilst the temperature still remained subnormal.

(6) At the onset there was vomiting and diarrhœa.

(7) Resection of the bowel and lateral anastomosis was decided upon as the obstruction was high up in the small bowel and had not been present very long.

(8) The vomiting was not of the so-called fæcal type.

(9) The appendix was normal in appearance, but a previous attack may have accounted for the omental adhesion.

Case II.—J.S., aged 41 years, a labourer, was admitted on January 18, 1920, complaining of pain in the abdomen and inability to take food. His previous health had been good, except for an attack similar to the present one five years ago.

Present Illness.—Nine days ago, whilst working in the country, he noticed "something heavy in the bowels low down, with pain in the left side." He was constipated at the time. Under medicinal treatment the bowels opened with enemata five days ago; since then there has been no action of the bowels. All the food taken has been vomited. He has lost much weight during the last week.

Patient was emaciated. He had an anxious expression and drawn features. His mentality was low. The abdomen was very distended and coils of intestine were visible through the abdominal wall and gave the abdomen a "ladder pattern." Nothing abnormal was felt in the rectum, but a mass was felt in the pelvis, which reduced in size after catherization of the bladder.

A median incision was made below the umbilicus and on opening the abdomen a large portion of the small bowel was found greatly distended and inflamed. The distended bowel was pulled out of the abdomen and a thin band of omentum was found near the ileo-caecal junction constricting the bowel. The band was easily broken with the fingers. The bowel was so distended that it was almost impossible to replace it in the abdomen, so a small incision was made into a distended portion and the bowel emptied into a kidney tray. The opening was closed with a purse-string suture and the portion thoroughly washed with saline solution and replaced. The abdomen was closed without drainage. Convalescence was slow.

The patient developed acute mania on January 21, 1920, and acute retention of urine on January 27, 1920.

His mental condition was unsound for some weeks. He was finally discharged quite well on March 18, 1920. In reference to his mental condition his relatives volunteered the information that "he was always peculiar and had a fancy for politics."

Commentary.

(1) There had been no action of the bowels for five days prior to operation and marked wasting and the patient showed marked intestinal intoxication.

(2) Pain was not a marked symptom, but occurred in paroxysms.

(3) The obstruction had apparently been only partial for some days.

(4) The distended abdomen had the "ladder" appearance remarked on in the previous case.

(5) The temporary opening of the bowel was of considerable advantage, although the procedure is not free from risk.

(6) The mental condition of the patient after operation was probably due to intestinal intoxication.

(7) The appendix was normal, but there had probably been a previous attack of appendicitis.

Case III.—G.H., female, aged 28 years, married, with one child, was admitted, complaining of abdominal pain.

The family and social history did not contain anything of importance.

The patient's previous health has been good, except that she

had influenza six months ago and a similar attack of pain to the present one month ago.

Present Illness.—Eight days ago she had an attack of vomiting, with colicky pain in the lower part of the abdomen and another attack three days ago. The vomitus was yellow in colour. Since the first attack the pain has come on in paroxysms. There is a feeling of tightness and hardness and gradual swelling, which starts in the hypogastrium and extends to the left. After the spasm passes off the swelling disappears and the patient sometimes hears a "gurgle." The bowels have not been opened for eight days and the patient complains of "belching of wind" constantly. Enemata have been without result.

The patient looks well. There is no abdominal distension and there is only slight tenderness in the hypogastrium. The abdominal muscles are relaxed. Palpation at times produces distension apparently of the tranverse colon and a large swelling is then evident, starting from the right and extending to the left. No tumour is felt.

The vulva is red and tender to touch. The vagina is hot. The uterus is displaced to the right. There is tenderness in the vaginal fornix. No fæces were felt on rectal examination in the lower bowel.

A diagnosis of partial obstruction of the transverse colon was made. The abdomen was opened in the middle line below the umbilicus and later the incision was extended above the umbilicus. The large bowel was found to be much distended and inflamed and some adhesions in the left iliac region were separated. The transverse colon was brought out of the wound and a stricture due to a new growth was found about its middle and there were large glands in the transverse mesocolon. The bowel above and below the growth was distended. The colon was freed and the affected portion with all evident glands was removed, the bowel being divided by means of the cautery at a distance of 15 cm. on either side of the growth. The stumps of the divided bowel were buried by means of purse-string sutures. The caecum was mobilized and easily joined to the sigmoid by means of a wide lateral anastomosis.

With the exception of *Bacillus coli communis* infection of the wound, which rapidly healed, the patient made an uninterrupted recovery and left hospital on January 27, 1920. The bowels acted without trouble. The radiographer reported that some stasis was present in the ileum.

The patient presented herself at the Out-Patient Department six months after leaving hospital and stated that she had put on 12.7 kilograms in weight and that her bowels had acted in a normal fashion daily without aperients.

There was no evidence of metastasis or local recurrence.

Pathological report: Adeno-carcinoma.

Commentary.

(1) The patient was only 28 years of age.

(2) There was no history of trouble up till one month of entering hospital.

(3) The blind ends of the bowel above the anastomosis have not resulted in stagnant pools.

Reviews.

REGIONAL ANÆSTHESIA.

In France the phase of anæsthetic practice spoken of as regional has received a considerable amount of attention, according to Dr. Sherwood-Dunn, whose recently published book[1] has been introduced with a hope that his exposition of the subject might be received favourably by the medical profession in America. The subject appears to have been treated to but scant attention in the English-speaking world, the probable reasons for this being the toxicity of cocaine which was formerly used as the anæsthetic agent and the frequency of failure from faulty technique.

Any method offering facilities for securing anæsthesia throughout hazardous operations is always very acceptable to the anæsthetist. Regional anæsthesia appears to furnish help in such risks as involve the possibility of drowning by fluids from the chest (empyema, hydatid, etc) or by blood, blocking or filling the air passages, owing to the preservation of an effective coughing reflex and in certain cerebral and cerebellar tumours in which the increased intracranial pressure has so depressed the respiratory efficiency that a general anæsthetic, especially chloroform, would occasion its failure. Further advantages of the method over those of general anæsthesia claimed by its advocates are its greater safety, the mortality since replacement of cocaine being nil, and the greater freedom from shock and dangerous after effects. Over local anæsthesia the advantages claimed are that, with the exception of injecting the mesentery in abdominal operations, the surgical field is not invaded by the anæsthetist, while its range of employment is the same as that of general anæsthesia.

The technique is that of Professor Victor Pauchet, a résumé of whose writings, together with that of other exponents in France, constitute the substance of the volume. Pauchet, the leading exponent of the method, has revised and amplified the author's manuscript, so that it is brought quite up to date. Morphine pre-narcosis is always used and the anæsthetic agent elected is novocain (procain, neocain, etc.). Suitable syringes, needles and concentration of solutions employed are described.

The principle of regional anæsthesia is that of nerve blockage by perineural injection of the anæsthetic solutions, combined with adrenalin, in suitable concentrations, either at the origin or along the trunks of the nerve or nerves supplying the field of operation. Improved technique and practice in France has reduced the number of failures to effect complete anæsthesia to 5%. In such cases of failure ethyl chloride is used to induce narcosis.

The technique presents no difficulty in the majority of operation cases, ordinary care being exercised in locating the land marks for the nerves to be blocked. In some operation cases, however, rehearsal of the procedure on the cadaver is advised, for instance, such as those involving excision of the upper or lower jaws and of the tongue, which entails infiltration of the Gasserian ganglion through the *foramen ovale*, or around the first and second division of the fifth nerve through the orbit, or of the latter division through the *foramen rotundum*, etc. Success is proportional to the practice of the administrator, but the mental attitude of the patient is also an important factor.

Doubtless regional anæsthesia has the advantages claimed; retention of consciousness through an operation may be regarded as advantageous by some patients. In prolonged operations this would appear to be a questionable benefit, since, as the author points out, patients sometimes cry out owing to the great mental strain resulting from anxiety and weariness. To combat pain arising from prolonged operation on a hard operating table, padding the table is necessary. Especially for nervous sensitive patients the eyes should be closed and the ears stopped. Silence should be observed by those engaged at the operation and all surgical manipulations should be gentle, especially in the abdomen.

This work is excellently produced on good paper, with clear, well-spaced type. The abundance and excellence of the figures greatly aid clearness of description.

[1] Regional Anæsthesia (Victor Pauchet's Technique), by B. Sherwood-Dunn, M.D.; 1920. Philadelphia: F. A. Davis Company. Sydney: Angus & Robertson, Ltd.; Royal 8vo., pp. 294, with 224 figures in the text. Price, 21s.

The University of Sydney Medical Society held a Conversazione on November 12, 1920, in aid of the Anderson Stuart Memorial Fund (see *The Medical Journal of Australia*, October 23, 1920, page 399). There was a large gathering, including His Excellency the Governor of New South Wales and Dame Margaret Davidson, who were received by the Chancellor of the University and the Dean of the Faculty of Medicine, as well as the President of the students' society. Many lectures and demonstrations were held and numerous chemical and pathological exhibits were on view.

Professor Wood Jones desires it to be known that there is a sufficiency of "subjects" in the anatomical department of the University of Adelaide for medical graduates who wish to study during November and December.

The Medical Journal of Australia.

SATURDAY, NOVEMBER 20, 1920.

The Foundation of Prosperity.

History is full of evidence of economic failure resulting from pestilence and plagues. At almost every epoch we find that the nations have been compelled to divert an inordinate amount of energy and vast sums of money to cover up some of the devastating effects of disease. The oft quoted history of the Panama Canal is but a limited instance of the waste encountered when industrial effort is maintained without a preliminary eviction of disease. The cost of ill health to any community is so enormous that it seems incredible that any government is bold enough to ignore it in its budgets. There is much talk about the necessity of limiting the baneful effects of such scourges as syphilis and tuberculosis and indeed small sums of money are invested for the purpose of reducing the incapacity due to these causes. The individual, if he be wise, places a monetary value on his ability to work and insures himself against incapacitating illness. Governments, however, make little or no provision of this kind and in Australia, at all events, are unwilling to expend a reasonable premium for the purpose of rendering the people healthier and more capable of sustained industrial effort.

The Federal Council of the Australian Natives' Association has just determined to ask the Federal Government to take up the question of the improvement of the public health by instituting some comprehensive scheme of medical research. It is undeniable that our knowlelge to-day is insufficient to effect more than a relatively small reduction in the general morbidity and mortality. A few diseases can be attacked with a good prospect of early success. The great mass of disease, as it affects us to-day, cannot be eliminated with the means at our disposal. Medical research is often disappointing and always tedious for the onlooker. Years of patient work are needed before an important step onward is recorded. But unless an extensive and well co-ordinated effort be made, we cannot achieve anything. To subsidize medical research is to invest money in a sound undertaking which, sooner or later, must return interest out of all proportion to the capital expended. In other countries this fact has been admitted. When the National Health Insurance scheme was introduced in England, the Government recognized this principle and set aside the sum of one penny per insured person for the purpose of medical research. During the first two years this money was allowed to accumulate. In 1913 the Medical Research Committee was formed under the presidency of Lord Moulton, of Bank, with Dr. (now Sir Walter) Fletcher as Secretary. In the early days the organization was defective, largely owing to the fact that the money was being expended dole-wise. The Committee was divided in its opinions as to whether there should be decentralization or concentration. No one seemed to be fearless enough to risk failure in the endeavour to achieve signal success. The objective of any extensive scheme of this kind must be to enlist the services of the most capable men and women, to train them in the many branches of medical research and to institute as many co-ordinated campaigns as possible. While research was placed in the hands of young graduates holding scholarships or fellowships worth a mere pittance for two or three years, progress remained unattainable. Only a few enthusiasts were willing and able to continue this work beyond the stage of apprenticeship. Moreover, there were but two or three positions worth holding from the point of view of remuneration. One of the first acts of the Medical Research Committe was to create reasonably paid positions for a few tried workers. There were too few and the promise held out to likely trainees was too nebulous to be attractive. The Committee started badly by expending money on bricks and mortar instead of concentrating on the building of careers. Favourable and generous offers were thrust aside and the Committee exhibited a lack of enterprise which threatened to spell failure. The opportunity, however, was not long in coming and the indifferent start was soon converted into a magnificent achievement. The War Office and the whole British Government recognized early in the second half of 1914 that this organization could be utilized to immeasurable advantage for war purposes.

The question of careers and continuation of service did not enter. The whole of the medical profession was available and the Medical Research Committee was allowed to utilize the brains and energies of a veritable army of clinical and laboratory workers. Special committees were formed to attack specific problems for the Government. As the years rolled on and as the difficulties multiplied, the activities of the Medical Research Committee extended. Infective processes affecting troops in the field, tropical diseases, food problems, the medical aspects of flying, the question of hæmorrhage and surgical shock and many other subjects were attacked by groups of competent investigators. We need but to refer to the valuable collection of the publications of the Committee to substantiate the statement that the investment of the penny per insured person, together with the contributions from the other departments of the Government, has proved extremely profitable. Since the war ended, the Privy Council has taken over the Medical Research Committee. The future will reveal whether sufficient provision will be made to attract the best research workers. Unless the young man or woman realizes that research work embraces a good prospect, that there is a ladder to climb that is worth climbing, we cannot expect either to survive the period of training.

There is in Australia to-day no need for the erection of large institutes. The first steps should be to have a small but active committee, like the Medical Research Committee on the other side of the world, to co-ordinate research. Positions in clinical and laboratory research, graded according to the experience and ability of the holders, should be created in considerable numbers. The committee should have power to enlist the services of competent practitioners to organize special investigations and to supervise or guide groups of salaried workers. Existing university and other laboratories and the public hospitals should be utilized for this purpose. When it is shown that the accommodation is insufficient, new institutes should be built. The amount of money required for such a scheme would be considerable. It would, however, not be a gamble, but a sound investment. Within five or ten years Australia would have a small army of trained workers whose efforts would increase Australia's industrial output a hundredfold.

CARDIAC MURMURS.

In the present issue we publish an article by Dr. J. Macdonald Gill in which he again deals with his conception of the production of heart murmurs. During a period of over twelve years Dr. Gill has sought a better explanation than that usually held. It will be admitted by the majority of physicians that there is considerable justification for the refusal of the theory that heart murmurs are due to the flow of blood through a narrowed orifice from one cavity to another. While the frequency of a presystolic murmur audible at the apex of the heart existing without any stenosis of the mitral orifice and that of a "to and fro" murmur audible at the aortic area without any aortic regurgitation is probably small, the occurrence of these conditions is proven. It is therefore obvious that the presystolic murmur in the one case and the diastolic murmur in the other cannot be produced by the blood passing or leaking back through an altered orifice. It might be held that in these exceptional circumstances the "fluid vein" theory would not apply. But another fact is brought forward by Dr. Gill which is highly suggestive. It is that it is an extremely common occurrence for a systolic apical murmur to develope early in a first attack of rheumatism in children. In these cases the mitral valves are but little altered. There is certainly no structural change sufficient to give rise to a failure of the valve cusps to close for the purpose of preventing a backward flow of blood from the ventricle to the auricle. Moreover, the same thing applies to diastolic murmurs heard in children during a first attack of rheumatism. If it be conceded that the usual explanation fails in the instance of the common cardiac murmurs associated with first attacks of rheumatism and is obviously erroneous in certain rare heart affections, it must be granted that the acceptability of the hypothesis in general may be questioned. It would be unwise to burn our boats unless we can be satisfied that our new resting place will provide for all our needs. In other words, we should not deny the possibility of murmurs being produced by the constriction of a cardiac orifice or by the leakage backwards of blood through an orifice imperfectly guarded by valves unless and until another adequate explanation is offered. The association of mitral stenosis and a presystolic murmur is too great to be ignored, although mere association is not proof of cause and effect. Dr. Gill offers an alternative explanation. He does not claim that he has proved that his hypothesis is correct, but he argues on *a priori* grounds and claims consideration for it on the basis of probability. Throughout his six articles he has unfortunately not marshalled his facts and arguments with sufficient clarity and conciseness. In consequence he has been misunderstood and unfairly criticized by some, while others have brushed aside his ideas as largely imaginative. His position might have been much stronger to-day, had

he endeavoured to supplement his clinical observations by a series of carefully planned histological and experimental studies. The weakness of his argument is caused by the fact that he has to seek physiological and physical facts from the work of others.

The hypothesis put forward by Dr. Gill, if we understand him correctly, is that all murmurs are produced by certain vibrations of the smooth muscular tissue situated at the junction of the right auricle and the superior and inferior *venæ cavæ* and of the left auricle and the pulmonary veins. It is assumed that smooth muscular fibres similar to those of the auriculo-ventricular bundle exist in the human heart in these situations. These vibrations are continuous and quite independent of the contraction of the auricles which is effected by the striped muscular fibres. Under the influence of certain abnormal chemical agents, such as the hypothetical toxin of the rheumatic virus, these vibrations become audible. The existence of the vibrations, which are usually about eight per second, is demonstrated in the cardiographic tracings of affected hearts. Dr. Gill suggests that the intensification of the normal feeble vibrations occurs when a certain degree of tension of the auricular wall and of the auriculo-ventricular valves is present. The harmonics of the slow vibrations would be audible as a sound at certain points of the chest wall determined by the presence of a continuous conducting medium. The argument in favour of the murmurs being produced by these muscular vibrations appears to be that they can be demonstrated in all cases when a murmur is heard. It is further to be noted that there are apparently no awkward exceptions to be explained, such as occurs in connexion with the fluid vein hypothesis and the presystolic murmur without mitral stenosis.

The manner of conduction of the sound of the vibrations is of importance. In the case of the presystolic murmur audible at the site of the apex beat, the heart muscle has probably suffered most intensely in the auricles. If there be tension of the left auricle and of the mitral valve, the vibrations arising at the junction of the auricle and the pulmonary veins would be conducted partly along the column of blood filling the ventricle and partly along the wall of the left ventricle to the apex. The apex of the heart approaches the chest wall as the ventricles begin to contract. Before this takes place it is sufficiently close to the chest wall for the vibrations to be conveyed to the receiver or ear piece of the stethoscope. The murmur would in these circumstances be first heard when the auricle is sending a continuous volume of blood into the ventricle and when the apex is close to the chest wall. It would cease to be heard when the mitral valve cuts off the blood in the auricle from that in the ventricle, when the auricular wall becomes flaccid and when the apex of the heart recedes from the chest wall. These occurrences take place immediately before the first sound. The production of a systolic or diastolic murmur associated with mitral or aortic disease, with the various forms of anæmia, with adherent pericardium, with aneurysm and so forth can be explained in a similar manner. There is nothing impossible or even improbable in the hypothesis. If the facts be correct, the acceptance of the interpretation must depend on the observation of consistent coincidence of vibrations and murmurs conducted in the same direction for the same period of the cardiac cycle. The facts require corroboration. Others must be able to demonstrate the vibrations in every cardiographic tracing of an affected heart. The vibrations must appear in the tracings when the receivers are applied mechanically to a given area of the chest wall, as well as when the receiver is held by hand. The histological evidence of the smooth muscular fibres at the junction of the auricles and the *venæ cavæ* on the one side and the pulmonary veins on the other must be presented. In the last place, an endeavour should be made to produce a defect in the mitral valve cusps of some animal without damaging the heart wall. If this could be done successfully, there should be no difficulty in determining whether or not a defect of the valve suffices to produce a murmur. While more evidence is needed before Dr. Gill can claim that his explanation of murmurs is the true one, we must admit that his work is significant and very ingenious.

THE FUTURE OF THE NO. 4 AUSTRALIAN GENERAL HOSPITAL.

In reply to a question asked in the Legislative Assembly of New South Wales on November 11, 1920, the Minister for Public Health gave the following information concerning the future of the No. 4 Australian General Hospital at Randwick. Negotiations were proceeding between the Commonwealth and the State Governments with a view to the transfer of the Hospital to the latter. While the ground and original buildings were the property of the State, it was recognized that the old buildings which were formerly occupied by State children, were not suitable for hospital purposes. The Commonwealth Government had erected 34 wards, an orthopædic department, an X-ray department, an operating theatre, stores, dispensary, laundry and numerous other departments at the cost of about £100,000. The hospital, as it stood to-day, had accommodation for approximately 800 patients and about 50 nurses. A joint committee had been appointed for the purpose of arranging the transfer. The Minister stated that it was the intention of the State Government to use the property for hospital purposes.

Naval and Military.

APPOINTMENTS.

Citizen Naval Forces of the Commonwealth.
Royal Australian Naval Reserve.

Promotion—

Surgeon-Lieutenant-Commander Edward Walter Morris (retired list) is promoted to the rank of Surgeon Commander (retired list), dated 1st June, 1920.

OSLER MEMORIAL FUND.

We have to acknowledge with thanks one further contribution of £1 1s. from Dr. Gifford Croll, of Sherwood, Queensland. The total amount collected is therefore £32 11s.. This small amount has now been forwarded to Oxford as the Australian contribution to the memorial of a great teacher.

Abstracts from Current Medical Literature.

PATHOLOGY.

(158) Dural Endothelioma.

F. B. Mallory has made a careful study of the development of the *dura mater*, arachnoid mater and the perineurium of peripheral nerves and also of the tumours that arise from these structures (*Journ. of Med. Research*, March, 1920). He states that there is no dural endothelium and the inner surface of the *dura* is bare except for fibroblasts. The arachnoid differentiates early in embryonic life out of the mesenchyme surrounding the brain and cord and divides the *pia* from the *dura*. It separates here and there from the *pia* to form the subarachnoid space, which is lined by fibroblasts only. For a while the arachnoid remains closely attached to the *dura*, to the distribution of which it corresponds exactly. Then it separates completely from the *dura*, which it leaves bare. After this separation the cells on the outer surface of the arachnoid form a layer of endothelial-like cells, which are undifferentiated, but potential fibroblasts. The future growth and thickening of the arachnoid depend on these cells. With advancing age these surface cells multiply, usually not uniformly, but here and there in foci and form cellular masses or buds, which occasionally exhibit a concentric arrangement (whorl formation). These buds show a marked tendency to invade the *dura* and in time the cells secrete intercellular substances and in this way form the arachnoid villi (Pacchionian bodies). The growing cells of the arachnoid frequently give rise to tumours which go under various names: dural endothelioma, psammoma, fibroma, fibro-sarcoma. The best term for the entire group of tumours would seem to be "arachnoid fibro-blastoma." This tumour derives the blood vessels and connective tissue of its stroma from the *dura*. It can invade the *dura*, the skull and the soft parts outside, but it cannot pass through the arachnoid, from which it has arisen, and invade the brain. The perineurium is either an outgrowth of the arachnoid or more probably an analogous differentiation of the mesenchyme around the nerves. Tumours frequently arise from the perineurium, occasionally in great numbers and are known under various names: neurofibroma, false neuroma, *fibroma molluscum* and, when multiple, as von Recklinghausen's disease. They differ distinctly from the arachnoid fibro-blastomata in one respect: they show a marked tendency to invade the nerve tissue and occasionally to extend along it to such a degree as to form the so-called plexiform neuro-fibroma. These tumours should be grouped under the term "perineural fibro-blastoma," although their close relationship to the arachnoid fibro-blastoma is evident.

(159) The Leucocytic Reaction in Influenza.

E. C. Rosenow records the results of a series of experiments undertaken with the view to ascertain the leucocytic response in influenza and in pneumonia complicating influenza (*Journ Infect. Diseases*, June, 1920). Leucopenia has come to be regarded as an important aid in the diagnosis of influenza. The cause of the leucopenia has been the subject of much speculation. It is thought by many observers that an unknown virus produces the initial leucopenia and that this virus interferes with the leucocytic response when secondary infection by streptococci or pneumococci is believed to occur as pneumonia developes. The author's findings, however, indicate that the reduction in leucocytes following injection of material from patients with influenza may not be due to an unknown virus, but to peculiar properties of the streptococci or other bacteria at hand in this disease. These streptococci have the power by growth *in vitro* and *in vivo* to produce a soluble filterable substance. The filtrates from broth cultures and from influenzal lungs and sputum when applied intrathecally in guinea-pigs have the power to cause sharp reduction in leucocytes. This reduction has occurred with regularity only in the animals injected with influenza strains and not following injections of streptococci in like dosage from similar conditions showing leucocytosis, or type pneumococci. The author concludes that the leucopenia in influenzal infection in man may be due to peculiar properties of the bacteria, which are now generally regarded as secondary invaders and not to an unknown virus. He considers that a persistent, marked leucopenia, or an increasing leucopenia in influenza predisposes to influenzal pneumonia and in the latter indicates a bad prognosis.

(160) Streptococcus Hæmolyticus.

In the *Journal of Infectious Diseases* (February, 1920) David J. Davis records the results of further observations on the hæmolytic streptococci of the alimentary tract. He has previously shown that hæmolytic streptococci occur in the crypts of the faucial tonsils in nearly 100% of individuals. Large numbers of streptococci are therefore constantly passing down the œsophagus into the stomach. It was with the object of ascertaining the fate of these virulent hæmolytic streptococci in the gastro-intestinal canal that further investigations were carried out. The author found that hæmolytic streptococci do not occur normally in any appreciable numbers in the gastro-intestinal canals of rabbits. When introduced into the stomach of the rabbit in large numbers, virulent hæmolytic streptococci may occasionally pass through the canal and appear in the fæces. Examination of the gastro-intestinal canal at various levels in rabbits thus fed indicates that hæmolytic streptococci do not develope appreciably in the intestines, nor do they readily gain a permanent foothold there. Rabbits with generalized streptococcus infections in joints, blood, etc., showed no hæmolytic streptococci in the intestinal contents. Gastric juice of normal acidity from man and from rabbits kills hæmolytic streptococci in from two to five minutes. Gastric juice in achylia may not kill them in several hours. In normal human fæces hæmolytic streptococci were not found in any of the 53 cases examined. Hæmolytic streptococci, when mixed with normal human fæces, will live in the ice-box for at least several days. In the incubator they tend to die out rapidly, probably owing to overgrowth and destruction by normal fæcal organisms.

(161) Green-Producing Cocci of Influenza.

Ruth Tunnicliff (*Journ. of Infect. Dis.*, May, 1920) has investigated the characters of *Streptococcus viridans* associated with influenza. The coccus is oftener lanceolate than round, generally it possesses a capsule and produces large, moist green colonies on blood-agar plates. It is, as a rule, insoluble in bile and rarely ferments inulin. The author has isolated this coccus from the œdematous brain in ten cases of influenzal broncho-pneumonia. In the majority they were grown in pure culture. In no instance was the Pfeiffer bacillus cultivated from the brain. The serum of rabbits immunized with strains of this coccus from influenza and its complications contained opsonins and agglutinins for other similar bile insoluble influenzal cocci and also for certain influenzal cocci, which were bile soluble and agglutinable by anti-pneumococcus sera. These results indicate that the green-producing influenzal cocci form a group, the members of which are closely related, as tested by their immunological characters.

(162) Adenomata of the Carotid Gland.

In the *Bulletin of the Johns Hopkins Hospital* (June, 1920) three cases of adenoma of the carotid gland are reported by Mont. R. Reid. The author regards this slow-growing pigmented tumour of the carotid gland, described as "carotid body tumour," as a simple hyperplasia of a part or all of the normal gland. It should, he thinks, be designated as an adenoma or hyperplasia of the carotid body. "Potato tumours," as described by Hutchinson and Gilford, do not come within the scope of the accepted meaning of "carotid body tumours." A "potato tumour of the neck" may be a primary sarcoma or carcinoma of the carotid body. Yellowish pigmentation of the body, flushing of the head and neck and a slow, somewhat apathetic state of mind may result from hyperplasia of the carotid gland.

PÆDIATRICS.

(163) Mercurial Preparations in Congenital Syphilis.

The treatment of syphilis with the different mercurial preparations is usually carried out in a haphazard

manner, the rule being to give as much mercury as the patient will tolerate without salivation or diarrhœa. Assuming that the amount of mercury eliminated in the urine in a given time would give an approximate index of the amount in the circulation, Ramsay and Ziegler made some experiments, the report of which appeared in the *Amer. Journ. of Dis. of Children* (November, 1918). From this the following conclusions could be drawn. Mercury, when given by the mouth, by inunction or intramuscularly to infants and children, is excreted at least partly by the urine. Mercurial ointment placed in contact with the skin of infants and children, even without any friction being used, is absorbed through the skin and excreted in the urine. It continues to be excreted in the urine for a variable time after all treatment has been discontinued. In these experiments precautions were taken to prevent the inhalation of mercury vapour. The area of skin to which the ointment was applied, was covered by wax paper, sealed all round. Mercury applied by inunction is readily taken up by the skin and eliminated in the urine and continues to be eliminated for a considerable time. When one inunction is given the maximum daily amount of mercury is usually eliminated during the following 24 hours, smaller amounts being eliminated for a variable time. An accumulation of mercury in the system follows continuous inunctions. Considerable amounts are eliminated from time to time, while in the intervals only traces are excreted. Mercury salicylate suspended in oil and given subcutaneously continues to be eliminated in the urine in appreciable amounts for 8 days or longer, the daily amounts varying widely. Mercuric chloride injected intramuscularly continues to be eliminated for 8 days or longer. In all cases in which mercuric chloride was used by mouth or intramuscularly, protein was found in the urine.

Working on these lines W. R. Ramsay and O. A. Groebner (*Amer. Journ. of Dis. of Children*, September, 1920) performed a new series of experiments. They have found that 50% mercurial ointments are to be preferred to the less concentrated forms and need not be repeated more often than twice a week instead of daily. The quantity of mercury absorbed is much increased by friction. Calomel ointment is absorbed but less rapidly and to a less extent than the mercurial ointment and should therefore be given in greater concentration. Salicylate of mercury in oil should be given twice weekly. Mercuric chloride by hypodermic injection, although the dose be very small, continues to be eliminated for six or seven days. The fact, however, that its use is frequently followed by the appearance of protein in the urine should exclude it from the treatment of syphilis in children. Calomel by the mouth is absorbed in small amounts and continues to be eliminated for a considerable time, so that it should be sufficient to give it at intervals of several days to avoid diarrhœa. Grey powder is absorbed to a small degree and eliminated rather rapidly. Large doses repeated daily would probably be necessary to maintain mercury in the circulation.

(164) Immunization Against Diphtheria.

Since the introduction of the Schick test, it has been possible to obtain a very accurate measure of the body's antitoxic immunity and consequently to determine the value of any new form of prophylactic treatment. For five years J. Blum (*Amer. Journ. of Dis. of Children*, July, 1920) employed the toxin-antitoxin method of active immunization and controlled it by the Schick test in an infants' home. He states that the Schick tests made on 1,076 children in an institution during a period of five years show that a failure to respond is evidence of the presence of sufficient antitoxin in the tissues to protect the the individual against diphtheria infection. Moderate or slightly positive reactions denote an insufficient amount of antitoxin in the tissues to protect the individual. When the reaction is very faint, there is probably sufficient antitoxin to protect. It is possible that the transfer of virulent organisms to these individuals might cause them not only to become carriers, but to suffer from a mild infection without any constitutional symptoms. Tests carried out on the members of 72 families showed that all the children in one family usually responded in the same way. In those instances in which divergencies occurred, the younger members usually reacted, while the older members failed to react. Infants under six months of age, because of the transmitted immunity of the mother, were exceptions to this rule. The appearance of a positive reaction in children up to three years of age who had previously failed to react, were found to be sufficiently numerous to justify retesting during this period at intervals of a few months, especially in infant asylums. This change is attributed to loss of inherited immunity. The full duration of the active immunity conferred on individuals susceptible to diphtheria by the injection of toxin-antitoxin is not yet ascertained. In one series of cases the immunity persisted in 100% of a group of 50 children for at least 22 months. Others have found that the immunity once acquired lasts for at least four and a half years. It is possible to render all the children in an infant asylum or similar institution free from diphtheria by injecting all the susceptible children with toxin-antitoxin mixture.

(165) Fluid Injections in Dehydrated Infants.

After experiments on rabbits, with reference to the intra-peritoneal method of injection of fluids, S. McLean and C. A. Lang (*Amer. Journ. of Dis. of Children*, May, 1920) decided to use this method in dehydrated infants. Of 269 injections of fluid into 76 infants, 92 were intra-peritoneal, 22 were sinus injections and 155 were subcutaneous. The mortality in the 76 cases was 56.5%. The amount of the subcutaneous injections varied between 90 c.cm. and 150 c.cm., in accordance with the size and condition of the child. In a few cases 0.00006 to 0.00012 grm. of atropine was added. On one occasion 120 c.cm. of a 6% dextrose solution was used. The solutions used for intra-peritoneal injection were of various formulæ and in quantity between 150 and 240 c.cm.. They were: 6% dextrose in normal saline solution; 6% dextrose in distilled water; normal saline solution; 2% sodium bicarbonate, with 2% dextrose solution; 1% sodium bicarbonate, with 1% dextrose solution. The authors summarize their results as follows: (*i.*) Injection of fluid into the peritoneal cavity in dehydrated infants is a simple method of procedure and gives no undesirable effects. (*ii.*) The size of the infant is no bar to its ability to utilize injected fluids. (*iii.*) From the results of injections in very small infants, weighing less than 3,000 grammes, it would appear that larger injections might be given to larger infants. (*iv.*) Age has no bearing on the infant's ability to utilize injected fluids. (*v.*) The pulse is more frequently affected after sinus and intra-peritoneal injections than after hyperdermoclyses. (*vi.*) The respiratory rate is more frequently affected by peritoneal injections than by sinus injections and hypodermoclyses. (*vii.*) The temperature is more frequently elevated in sinus injections than in the others. (*viii.*) Weight gains were more frequently noted following intra-peritoneal injections. (*ix.*) Certain infants do not show any improvement until they have had repeated injections of fluid. (*x.*) The shorter the interval between the onset of symptoms and the beginning of treatment, the greater the response.

(166) Artificial Infant Feeding.

M. Cotheuiner (*Amer. Journ. of Dis. of Children*, May, 1920) questions the advisability of following the older methods of infant feeding, as embodied in the modern text-books. The tables show that a new-born baby should only be given 30 c.cm. of food every two hours and the older infant is too often fed on large quantities of a dilute food, when a smaller, more concentrated food would suffice. The infant should be given enough food in sufficient concentration and the food should be continually increased, both in quantity and quality as the baby shows himself able to deal with it. When four or more opposing teeth appear, the infant is physiologically ready for some semi-solid food. At the age of nine or ten months the baby ought to be ready and able to digest undiluted cow's milk; shortly after he should be given cereals, strained at first, potatoes, baked and mashed well, stale bread, toast, stewed fruits well mashed and even an egg. To sum up, sufficient cow's milk mixture should be given at each feeding and an increase made in quantity and quality, as required by the infant. Whole milk should be given at from eight to twelve months. Semi-solid food should be given as soon as four or more opposing teeth appear.

THE INFLUENZA EPIDEMIC OF 1919.

(*Continued from Page 445.*)

III.—Methods of Control.

In his account of the alleged origin and assumed manner of spread of the disease, Dr. Armstrong states that the Commonwealth Quarantine kept it at bay for a period of nearly three months, notwithstanding the fact that 326 persons are said to have suffered from influenza within the quarantine stations. The New South Wales Department of Public Health regarded the discharge of persons after an attack, still harbouring the bacillus of Pfeiffer, with apprehension. These so-called "carriers" were closely watched, but it soon transpired that no infection spread from them. Long before the disease manifested itself in the State of New South Wales plans were laid for meeting an outbreak, should it occur. The now historical resolutions of the conference of November 26 and 27, 1918, are reproduced. Dr. Armstrong gives reasons for the action taken by his Department in regard to traffic with the neighbouring infected State and to quarantine arrangements at the border. No doubt the initial fault lay with the Victorian Health Department in delaying to notify the Director of Quarantine of the presence of infection within the State. It is held by many that this failure did not justify the New South Wales Department of Public Health in failing to carry out its undertakings as set out in the fifth resolution of the conference. This incident, however, has but a small bearing on the question of the value of the measures adopted to check the spread of the epidemic. Dr. Armstrong gives a very interesting account of these measures. It is unnecessary to refer in this place to the organization, committees, councils and other bodies created for the purpose of combating the infection. Everyone concerned undoubtedly put much energy into the campaign and an immense amount of work was accomplished. We are told that the poor received all the aid that could be extended to them, that the several agencies succeeded in providing succour to those in need and that the disastrous epidemic was handled in a manner calculated to mitigate the ill effects as much as possible. That a state of panic was produced is admitted. That the daily press and possibly some medical practitioners were responsible for some measure of this panic is not admitted by Dr. Armstrong. We cannot refrain from the opinion that the inordinate amount of publicity given to the development of the epidemic resulted in a public scare, which apparently tends to lower resistance and to favour the spread of an infection. Should Australia again be visited by a serious infective disease in pandemic form, we trust that the health authorities and those assisting them will be allowed to work silently and without embarrassing publicity.

The measures adopted are summarized in a table under ten headings. The first is State quarantine, including inter-State and intra-State restrictions of travel. Dr. Armstrong states that any good results of either inter-State or intra-State quarantine must have been very meagre. He admits that it was too late to apply inter-State quarantine after the first case had been recognized and he appears to be very doubtful whether land quarantine could have any value in so elusive and subtle a disease as influenza.

The provision of hospital accommodation is the second measure mentioned, while the provision of medical and nursing services in the homes of the sick is the third. Dr. Armstrong wisely dismisses these necessary expedients with the sentence: that no one would question their utility. In a previous part of the chapter he has described in considerable detail the organization of the 25 influenza hospitals and of the medical and nursing service organizations. He also passes over the fifth measure, namely the distribution of food, drugs, blankets, clothing and money to influenza patients in need of such assistance.

The fifth measure is compulsory notification. As has already been stated, "pneumonic influenza" was proclaimed a notifiable disease and uncomplicated influenza was notifiable only at the request of the Department. It is therefore obvious that the information gained from notification was not a reliable guide to the extent of the infection. Dr. Armstrong states that it had but a limited value. He regards it as having been useful during the early weeks of the epidemic, because it provided the health authorities with information concerning local centres of infection, often at an early date. Later, when the epidemic had gained a firm hold, it became useless.

The sixth means comprised the compulsory isolation of patients and those who had been in contact with patients. Dr. Armstrong is of opinion that this measure was of great importance in the early stages, but that it ceased to have any value at a later stage. Moreover, he is inclined to attribute to it a disadvantageous effect, inasmuch as neighbours refused to enter a house in which there were isolated patients, to render assistance. He has reason to state that isolation proved most valuable in sparsely populated rural districts.

The seventh measure of prophylaxis is a logical corollary to the accepted assumption that the infection of influenza is transmitted by means of sprayed droplets in sneezing, coughing and talking. In these circumstances dissemination would naturally occur when people congregate together in large numbers. Dr Armstrong refers to the large number of persons who were attacked by influenza about forty-eight hours after attending the Peace Ball on June 5, 1919. Dr. Armstrong is therefore on safe ground when he applauds the action of the Government in restricting assemblies of persons during the period of the epidemic. In regard to schools, he expresses the view that the closure of primary schools in the cities does more harm than good. We have frequently pointed out in these columns the danger of allowing children who may be passing through the incubation of an infectious disease, to frequent the gutter and other favoured playgrounds of the young. On the other hand, he recommends the closing of country schools during the currency of an epidemic.

The eighth expedient in the attempt to combat influenza has already given rise to much controversy of a varied and not always dignified character. The expedient is masking. Dr. Armstrong deals very temperately with this dangerous subject. He admits that the evidence for or against the value of masking is difficult to obtain. He states that nearly all the medical officers who served in the influenza hospitals and the majority of medical practitioners hold the view that the use of masks among those in attendance on patients was an invaluable safeguard. He himself holds this opinion very strongly and is convinced that, had the expedient not been adopted in the sick-room or clinic, the number of nurses and doctors attacked by the disease would have been very much greater. He is willing to concede that masking in the open air during the first three weeks of the epidemic was a mistake. On the other hand, he upholds the enforcement of masking in closed tram-cars, railway-carriages, public lifts and shops and the like. He states that the evidence available is not strong enough to warrant the compulsory enforcement of masking, but he advocates its use by persons exposed to infection during an epidemic. He denies that the wearing of a gauze mask can exercise any deleterious effects on the wearer and in support of this belief he states that the health of persons engaged in hospitals was not injured by the wearing of masks. It appears to us that the argument is not a very sound one. Moreover, Dr. Armstrong has apparently overlooked two very important practical points connected with this expedient. The first is that the great majority of persons who wore masks under compulsion, did so in such a manner that no protection could have been afforded. The second fact which was frequently noted during the epidemic, was that the gauze or lint placed in front of people's mouths and noses was frequently begrimmed with dust and dirt. From an hygienic point of view this aspect should not be ignored.

In the ninth place, the health authorities employed for a considerable time the spraying of a 1% solution of zinc sulphate for the purpose of inhalation. As is well known, this practice was discontinued after it had been shown that benefit did not result from its application.

The last measure of defence mentioned is prophylactic inoculation. Dr. Armstrong states that much consideration was given to the question of inoculation with vaccines as a prophylactic measure before the outbreak of the disease. At the Melbourne Conference it was determined that public vaccine depôts should be established. He points out that no vaccine could be expected to protect against the primary.

infection of influenza. It is obvious that this is so, in view of the fact that the causal organism of the disease is unknown. The reasons in favour of applying inoculation were that it was hoped to lessen the incidence and severity of complications and thus to reduce the mortality. Dr. Armstrong gives much information concerning the methods adopted in carrying out vaccination. He shows that the composition of the vaccines prepared in Sydney differed materially from those prepared at the Commonwealth Serum Laboratories. The number of bacteria contained in each cubic centimetre was far greater in the Sydney than in the Commonwealth preparations. It appears also that a considerable variation in dosage existed according to the amount of vaccine available. Free inoculation depôts were opened and the number of persons who rushed to these depôts, was very large. During the first week a record was kept of the name, age and sex of each person vaccinated. Later the inoculation was carried out at such high pressure that the only fact recorded was the number of inoculations. Dr. Armstrong says that inoculation became exceedingly popular. Many persons believed that inoculation had a beneficial effect on chronic catarrhs and rheumatoid arthritis. It was not uncommon for a person to submit himself for re-inoculation every two or three weeks. Free inoculation was offered from November 27, 1918, to May 17, 1919. Although there were 1,265 depôts in Sydney and its suburbs, the greatest number of three-hour sessions held on any one day was 88. Up to January 26, 1919, 69,683 persons were inoculated. From this time up to the time of the closing of the depôts, at least 375,000 inoculations were undertaken in the Sydney depôts. Dr. Armstrong estimates the number of inoculations in the public depôts in country districts at 225,000 and by private practitioners at 150,000. This brings the total number of inoculations to approximately 819,683. Dr. Armstrong is inclined to the belief that the number of persons involved was 404,843.

Information was gained from the patients treated in the various hospitals in the metropolis concerning the number of inoculations, if any, the dates of inoculation, the date of onset and admission and other material details. We note, however, that the information concerning inoculation was derived from the patients' statements. The records deal with 12,786 cases, including 814 who were members of hospitals' staffs. For the purpose of estimating the value of inoculation in reducing mortality, the members of the hospitals' staffs have been excluded. This was done firstly because no selection was exercised as was the case with the ordinary patient. Of the latter, only those suffering from the severer forms of influenza were admitted to hospital. In the next place, it was recognized that the early diagnosis and admission in the case of members of a hospital staff materially improved their chances of recovery. Of 11,972 patients, 4,155 had been previously inoculated, while 6,249 had not been inoculated. The mortality among the inoculated was 10.7% and among the uninoculated it was 16.5%. The mortality among those inoculated three times was 8.2%, as compared with 11.1% among those inoculated once only. The mortality among the 1,568 patients from whom no information concerning inoculation could be obtained, was 26.5%.

Information is given concerning 752 members of the hospital staffs who had been inoculated and who subsequently contracted influenza. In 68 instances the date of inoculation was not ascertained. Two of the patients were attacked by influenza on the day following their last inoculation, six on the second day, seven on the third day, six on the fourth day, nine on the fifth day, seven on the sixth day and so on. Over 500 were attacked later than 28 days after the date of the last inoculation. These figures are quoted in refutation of the suggestion that inoculation might produce a negative phase during which persons would be more susceptible to influenza infection.

In reviewing the information collected with great pains and considerable care, Dr. Armstrong finds that inoculation reduced the severity of the attacks and diminished the mortality. From the reports received from medical practitioners throughout the State, it would seem that the majority of them gained the impression that inoculation modified the severity of the attack. Dr. Armstrong points out that its power to prevent complications was not infallible. It will be remembered that the vaccines were prepared during a period of excitement. There is little doubt concerning the origin of the influenza bacillus used for the vaccines. Even if there be many types with varying virulence or pathogenicity, it may be assumed that the majority of the vaccines contained members of those strains which were associated with the illness actually present in Australia at the time. We learn from Professor Cleland's contribution that in eight cases only out of a total of 117 influenza bacilli were found in the lung associated with streptococci, staphylococci or yeast and in 29 further cases associated with pneumococci. It is doubtful whether fatal pulmonary complications are attributable to the influenza bacillus and consequently, the significance of this bacillus in a vaccine would appear to be small. In regard to the pneumococci no endeavours to ascertain the types involved were possible during the period when the vaccines were being prepared on a large scale. It is not improbable that the value of influenza vaccine depended on the inclusion of pneumococci of the type that was producing grave lesions. The unreliability of the vaccine to prevent pulmonary complications may have been due to the haphazard inclusion of all sorts and conditions of pneumococci at hand. In the Sydney vaccine was included a *Staphylococcus aureus*. Surely that was unnecessary ballast. No one has yet thrown light on the rôle played in the pathology of the disease by the streptococci. Their inclusion in the vaccine was at best an aim in the dark. The fifth ingredient in the Sydney vaccine was a Gram-negative micrococcus, while the fifth ingredient of the Federal Serum Laboratories' vaccine was a Gram-positive diplococcus. We must assume either that these constituents of the vaccines were of no prophylactic moment or that the one or the other vaccine was deficient in an important principle. These considerations appear to us to indicate that the small measure of apparent success resulting from vaccination was a chance result of an unscientific mixture of bacteria. We hope that something better will be available before the next severe epidemic of influenza appears. The influenza vaccine of 1918-1919 is a very insecure reed on which to rest the safety of many lives.

(To be Continued.)

British Medical Association News.

SCIENTIFIC.

A meeting of the New South Wales Branch was held at the B.M.A. Buildings, 30-34 Elizabeth Street, Sydney, on September 24, 1920, Dr. C. Bickerton Blackburn, O.B.E., the President, in the chair.

Dr. J. Macdonald Gill read a paper on heart action and murmurs (see page 469) and demonstrated a number of cardiographic tracings on the screen. Several of these tracings have been published in *The Medical Journal of Australia* (November 15, 1919, pages 413-414), while others have been published in the *Australasian Medical Gazette* (March, 1909, pages 126-127, and January, 1911, pages 2-9).

Professor A. E. Mills referred to the fact that all the tracings showed the small vibrations when the receiver was applied over the cardiac area. He had not been able to gather what Dr. Gill regarded as the significance of these vibrations. Before endeavouring to deal with the subject of the paper, he wished to ask Dr. Macdonald Gill to amplify his remarks in certain directions.

Dr. Macdonald Gill stated that the tracings were selected for the purpose of illustrating the points with which he was dealing. He held that the vibrations were manifestations of fibrillation of the auricles. The contraction of the auricles was shown by the absence of the "*a*" wave of the venous pulse and by the irregularity of the venous pulse. It was frequently held that a presystolic murmur disappeared when auriculaar fibrillation set in. He claimed that there were exceptions to this. The origin of his conception of the causation of murmurs was the observation of vibrations in a case of heart block. As he was at that time unable to account for these vibrations, he had kept a look out for them in other cases and had found them in connexion with a large number of conditions. He stated that in all

cases of recent rheumatic heart affection the vibrations were obtainable. According to his conception, a presystolic murmur was not necessarily due to mitral stenosis. The murmur was produced in the auricular muscle.

Professor Mills thanked Dr. Gill for his explanation. According to the tracings shown by Dr. Gill, small waves, evidently due to vibrations, were present in all of them, but the interpretation of them was a difficult matter. It was a curious fact that in the thousands of tracings made by Mackenzie and Lewis there was no evidence of such small waves as were shown in Dr. Gill's tracings. This seemed to him to be a very important fact. As to the assumption by Dr. Gill that these waves, undoubtedly due to vibration, were the cause of presystolic murmurs and that they became audible when there was sufficient tension of the valves, he could only say that he could not agree with it. He was satisfied that in the vast majority of cases presystolic murmurs were associated with stenosis and that the murmurs were dependent upon the stenosis. He could throw no light on the causation of the little waves which Dr. Gill had recorded in his tracings.

Professor Mills stated that there was another aspect of the question. Assuming, as he was bound to do, that these presystolic murmurs were bound up wtih the stenosis, he asked whether they told them anything else. They did not yield any information about the gravity of the lesion; they did not indicate to what extent the heart was crippled by the lesion. They were forced to look to other signs and symptoms for this information. Generally speaking, the size of the heart told them more as to the extent of the damage to the valves than anything else, but it was necessary to exercise caution in gauging the size of the heart by percussion, because emphysema might mask the size of the heart and lead to a very erroneous conclusion. There seemed to him to be a belief that dilatation of the heart was a bad thing. He looked upon it as a physiological phenomenon and as part of a necessary compensatory mechanism. It occurred in a healthy boy who ran a race or indulged in other violent exercise. In the later stages of mitral disease dilatation had an evil significance and with regard to hypertrophy it must be ever borne in mind that this was dependent upon a good blood supply through the coronary arteries. It must also be remembered that in a great number of valvular diseases syphilitic affections and other degenerative changes of the coronary arteries determine whether there would be much or little hypertrophy.

Professor Mills dealt in some detail with the question of raised blood pressure. He warned his audience against the harm done by telling a patient that he had increased blood pressure. The blood pressure in healthy individuals was not always at a fixed level. It was not always easy to determine when a blood pressure slightly above the standard was an index of a functional disability. He held that it was unnecessary to forbid a patient to do all those things he wished to do merely because his blood pressure reading was higher than the standard. Practitioners often forgot that the systolic blood pressure reading simply told them of the force of the heart. The diastolic pressure was entirely independent of the heart and was governed by the vaso-motor tone and the condition of the arteries. Not infrequently the diastolic blood pressure did not fall until death was about to take place, although signs of cardiac failure had existed for some time. In conclusion, Professor Mills referred to a patient whom he had had under treatment. This man's systolic and diastolic blood pressures were falling and the outlook appeared to be very grave. After a time, however, the diastolic pressure started to rise; the systolic pressure followed suit and then the patient regained moderately good health. Professor Mills thought it was unwise and unnecessary under certain conditions to endeavour to reduce a high blood pressure.

Dr. George E. Rennie stated that he had listened with great interest to Dr. Gill's paper. He was, however, not convinced of the truth of his explanation. Perhaps he had not grasped the full meaning of Dr. Gill's views. Dr. Gill had ascertained the existence of the vibrations that were graphically depicted in the tracings. If these vibrations were the cause of the murmur, how did he account for variations in the murmur? Sir James Mackenzie, Lewis and other exponents of what might be called the new cardiology, had got away from the old ideas. He was inclined to think, however, that the older observers were not so far wrong as Mackenzie had assumed. He himself had for long held that murmurs were of small importance. Irregularity of rhythm was often of greater importance because it indicated a disordered myocardium. Dr. Rennie referred to cases in which the double murmur of aortic disease was audible in the absence of aortic regurgitation. If there were no cardiac hypertrophy, the diastolic murmur could not be an indication of aortic regurgitation. After discussing these cases from a clinical and pathological point of view, he arrived at the conclusion that they still needed an acceptable explanation of the "to and fro" murmur without symptoms.

In the next place, he spoke of presystolic murmurs in persons with adherent pericardium. It was possible to confuse a friction sound with a presystolic murmur due to mitral stenosis. A presystolic murmur without signs of hypertrophy of the right ventricle should make them suspicious of some extra-cardial causation of the murmur. He thought that the time had arrived when practitioners should realize that there was no justification for treating every case of heart disease with digitalis. The use of digitalis was very limited. Only in certain cases of heart failure should this drug be employed.

Dr. Sinclair Gillies stated that he had experienced difficulty in following Dr. Gill's explanation of the causation of cardiac murmurs. It seemed to him that it would be easier to accept the old explanation of the sound of a murmur being produced by the movement of the fluid than that of the sound being due to contraction of the auricular muscle. He asked Dr. Gill whether he did not admit that leakage of blood through the valves had something to do with the production of murmurs. He claimed that it was difficult to understand how the sound could be produced by the contraction of the auricular muscle. Dr. Gillies called attention to the rate of the vibrations which Dr. Gill had said were 8 per second. Muscular tremors usually took place at about this rate. He suggested that the vibrations seen in the tracings might be due to muscular vibrations on the part of the observer. If they were produced in the heart, they should be capable of demonstration in the electro-cardiagram. He found that the whole subject of the production of murmurs was very involved. He was not prepared entirely to disregard cardiac murmurs from the clinical point of view. He held that the pendulum had swung too far in the other direction. It could be stated broadly that a person with an organic murmur did not live as long as a person without a murmur.

In dealing with the question of blood pressure, Dr. Gillies stated that he agreed in the main with the views expressed by Professor Mills. He was not prepared to leave all cases of increased blood pressure untreated. In many cases it was impossible or inadvisable to reduce the blood pressure, but in other cases this could be effected with considerable benefit. Accurate readings were impossible when there was marked intermission of the heart beat. He condemned the practice of drawing far-reaching conclusions from a single reading of the blood pressure. He agreed that irregularity of the pulse was of more value than the presence of a murmur in estimating the condition of the cardiac murmur, but warned practitioners against giving undue prominence to either irregularity or intermittence. In a large majority of cases iregularity was due to reflex or toxic causes and did not connote diseased heart muscle. In regard to prognosis, he maintained that *pulsus alternans* was probably the only form of intermission which was of serious importance.

Dr. W. F. Litchfield dealt at considerable length with the causes of heart failure in cases of œdema of the lungs and of asphyxia. After having described the condition of the lungs and of the circulation in death from drowning, he passed on to the consideration of heart failure due to diphtheria and that due to rheumatism. In the rheumatic heart the muscle failed because it was unable to deal with its load. In the post-diphtheritic heart the organ failed because its conducting power was damaged. In this instance the driving power was quite equal to its load. In the rheumatic heart there was often not enough mechanical damage to account for the hypertrophy present. Dr. Litchfield discussed the suggestion that the rheumatic

poison stimulated the muscular tissue of the heart and thus led to its hypertrophy. This was a very ingenious theory, but it had one serious defect. If hypertrophy were produced in this way, it would be reflected in the circulation. They had ample evidence that this was not so.

Dr. Litchfield thought that there was reason to refuse Dr. Gill's explanation concerning the production of murmurs. The common systolic murmur took place at a time when the right auricle was just beginning to go into diastole. It would therefore scarcely be in a condition to produce vibrations. The remainder of Dr. Litchfield's remarks had reference to the importance of the peripheral end of the circulation in conditions tending to cardiac failure.

Dr. R. Hamilton Marshall stated that he did not feel inclined to criticize Dr. Gill's paper. In all cases of heart disease, the important point to find out was the condition of the myocardium. He held that murmurs were useful in ascertaining the situation of a valvular lesion. In view of the fact that valvular lesions were able to cause degeneration of the myocardium, they were of great clinical importance. He regarded mitral stenosis as a very serious lesion. It was cicatricial in nature and was of slow onset. From the clinical point of view it was important to recognize that the various chambers of the heart were able to stand varying degrees of strain. The left auricle stood the strain badly and consequently obstructive lesions of the mitral valve readily produced dangerous symptoms. In ascertaining the condition of the myocardium, it was necessary to have regard to four conditions. In the first place, there was the quality of the heart sounds; the loss of tone was of great importance. In the second place, there was the rate of the heart beat. In the third place, the clinician should ascertain whether any of the many forms of arrhythmia were present. Dr. Marshall called attention to the fact that extra-systoles were of no importance. In the fourth and last place, the intensity of the second sound should be regarded as the indicator of the condition of the ventricular muscle. In connexion with the diagnosis of aortic disease, Dr. Marshall emphasized the necessity of a careful study of the pulse. Not only should its quality be noted, but also the condition of the vascular wall. He was in the habit of teaching students to think of the coronary artery when they were feeling the radial artery. Blockage of the coronary artery was followed by degeneration of the heart wall. As was well known, atheroma of the coronary artery in old people was frequently manifested by a systolic aortic murmur. In these cases sudden death was liable to occur.

Dr. F. Guy Griffiths said that he thought that Dr. Gill had a better case than he had presented that evening. He, the speaker, had had many opportunities of hearing Dr. Gill's views. While he was not prepared to go as far as Dr. Gill had gone, more especially in regard to the denial that the old view spoken of as the fluid vein theory never held good, he had to admit that Dr. Gill had very strong evidence that the old view did not explain all murmurs. In cardiograms of various observers, including Sir James Mackenzie, fine vibrations could be demonstrated. These vibrations must be produced by something. Mackenzie had not suggested any explanation, while others had shown that they were produced in the *sinus venosus*. There was no doubt concerning their reality. Having found these vibrations, Dr. Gill had wondered whether they might be the cause of murmurs and had been able to demonstrate that they corresponded to the occurrence of murmurs. He understood that Dr. Gill did not claim that he had proved his hypothesis. Further research was necessary and he, the speaker, considered that the evidence adduced up to the present demanded this further study. He thought that it was wise to mention that Sir James Mackenzie had not accepted Dr. Gill's explanation.

Dr. Harvey Sutton, O.B.E., called attention to the fact that the track of investigation had always been in the reverse direction to the blood current. The older observers had sought information regarding the cardio-vascular apparatus from the pulse. In their student days the present generation had concentrated on valves and the apex beat. The more recent observers had studied the auricle and the *sinus venosus*. He thought that greater attention should be paid to the study of the venous circulation. In referring to the prognosis of heart affections, Dr. Sutton remarked that approximately 1% of school children had some form of organic heart defect, either congenital defect or mitral endocarditis. Sutherland had held that the school medical officers were not justified in regarding mitral regurgitant bruits as of any real importance. He acknowledged the dangers of mitral stenosis as indicated by a presystolic murmur. Still had claimed that mitral lesions were rheumatic in origin and the mitral valvulitis one of the first manifestations of rheumatism. It had been ascertained that while 1% of school children had heart defects, the frequency of heart disease in school teachers was but 0.5%. Dr. Sutton suggested that this might mean that at least half of the children's hearts recovered completely or that the children were in the cemetery. He believed the latter. He was inclined to think that too much emphasis had been laid on the myocardial condition and not enough on the supply of the peripheral segment of the apparatus. He pointed out that the "heart" child was shorter and lighter than the normal child and was dull. He contrasted the "heart" child with the "thyreoid" child, who was bright, not dull. In his opinion, too much of their knowledge was gathered from the study of heart disease shortly before death. The study of the early stages was of great importance.

Dr. C. Bickerton Blackburn, O.B.E., congratulated Dr. Gill on his thoughtful work in connexion with heart murmurs. He was hardly convinced of the correctness of Dr. Gill's views. It was difficult to believe that the valves had nothing to do with the production of the murmurs. He asked why the presystolic murmur was nearly always associated with a contracted mitral valve. Again, in auricular fibrillation the murmur nearly always disappeared. He suggested that eight vibrations per second could not give rise to a musical sound. It appeared to him that the presystolic murmur heard in cases of adherent pericardium was always associated with myocardial changes. If the heart were dilated, there would be a relative alteration in the size of the orifices.

Dr. Blackburn agreed with Professor Mills that it was a great mistake to tell a patient what his blood pressure was. In dealing with the question of prognosis he regarded the size of the heart as of chief importance. When the blood pressure was high and the heart not enlarged, the condition was frequently of nervous origin and the prognosis was by no means bad. On the other hand, the association of a high blood pressure with a large heart was much more serious. He attributed little importance to systolic murmurs in children. Before forming an opinion in these cases, he would require to know the state of the heart. It was possible that the recovery of many of these children might account for the reduction of the frequency of cardiac defect of children as compared with adults. Many of the children with heart lesions did not live long enough to reach adolescence. It was often difficult to know the size of the heart in a patient affected with emphysema. X-ray examination was of great assistance in these cases.

In his reply, Dr. Macdonald Gill thanked Dr. Blackburn and others for their interesting remarks in discussing his paper. He stated that Mackenzie had not described the vibrations, but he claimed that that was no argument against their existence. He pointed out that it was 18 years before Mackenzie had accepted Lewis's views concerning auricular fibrillation. He stated that "to and fro" aortic murmurs were at times associated with normal valves. He referred to cases published by Finlayson and others. In reply to Dr. Gillies, he said that it was inconceivable that the vibrations were artefacts. He had shown that the vibrations usually occurred only when the receiver was applied to a small area of the chest wall to the left of the sternum. The tremors of *paralysis agitans* were about eight per second, but hysterical tremors were about 20 or more per second. In regard to the objection that during ventricular systole the auricles were flaccid and could therefore not give rise to a sound, he pointed out that in this period of the cardiac cycle the heart impinged on the chest wall and the vibrations which apparently were continuous, became audible as a murmuer, usually systolic.

The undermentioned have been elected members of the Victorian Branch:—

Ronald Cuttle, Esq., M.B., B.S., Melb. (1920), No. 11 Australian General Hospital, Caulfield.

Clifford Ellingworth, Esq., M.B., B.S., Melb. (1920), Box Hill.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

The undermentioned have been registered under the provisions of the *Medical Act, 1912 and 1915*, as duly qualified medical practitioners: —

Halkyard, Francis Percival, M.B., Bac. Surg., 1914 (Univ. Melbourne), Newcastle.
Hull, Walter John, M.B., Mast. Surg., 1920 (Univ. Sydney), Lewisham Hospital.
Iceton, Sydney George, M.B., Mast. Surg., 1920 (Univ. Sydney), Newcastle Hospital.
Sheppard, Edmund Macarthur, M.B., Mast. Surg., 1920 (Univ. Sydney).
Weaver, Ralph Edward, M.B., Mast. Surg., 1920 (Univ. Sydney), Lewisham Hospital.
Wyper, Jessie Jardine, M.B., Mast. Surg., 1920 (Univ. Sydney), Mosman.
Whitehead, Hebden, Hoffman, M.B., Bac. Surg., 1920 (Univ. Melbourne), Woodburn, N.S.W.

For additional registration:—

Jamieson, Hugh Hunter, F.R.C.S., Edin., 1920.
Willcocks, George Charles, M.R.C.P., Lond., 1919

Medical Appointments.

During the absence on leave of Dr. J. C. Catarinich (B.M.A.), Dr. D. D. Cade, D.S.O. (B.M.A.), has been appointed Acting Medical Superintendent of the Hospital for the Insane at Beechworth, Victoria.

Dr. Erwin Kelmar (B.M.A.) has been appointed Public Vaccinator at Dookie, Victoria.

The appointment of Dr. W. A. S. Bridgeford (B.M.A.) as District Medical Officer and Public Vaccinator at Broad Arrow, is announced in the *Government Gazette of Western Australia*.

The appointment is announced of Dr. Garnet Halloran (B.M.A.) as Honorary Ear, Nose and Throat Specialist to the Deaf, Dumb and Blind Institution of New South Wales.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser," page xxiii.

Broken Hill and District Hospital: Senior and Two Junior Resident Medical Officers.

Ballara Hospital, near Cloncurry, Queensland: Medical Officer.

Medical Appointments.

IMPORTANT NOTICE.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429 Strand, London, W.C..

Branch.	APPOINTMENTS.
NEW SOUTH WALES. (Hon. Sec., 30-34 Elizabeth Street, Sydney.)	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIA. (Hon Sec., Medical Society Hall, East Melbourne.)	All Institutes or Medical Dispensaries. Manchester Unity Independent Order of Oddfellows. Ancient Order of Foresters. Hibernian Australian Catholic Benefit Society. Grand United Order of Free Gardeners. Sons of Temperance. Order of St. Andrew. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND. (Hon. Sec., B.M.A. Building, Adelaide Street, Brisbane.)	Australian Natives' Association. Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIA. (Hon. Sec., 3 North Terrace, Adelaide.)	Contract Practice Appointments at Renmark. Contract Practice Appointments in South Australia.
WESTERN AUSTRALIA. (Hon. Sec., 6 Bank of New South Wales Chambers, St. George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND: WELLINGTON DIVISION. (Hon. Sec., Wellington.)	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

Nov. 23.—N.S.W. Branch, B.M.A.; Medical Politics Committee; Organization and Science Committee.
Nov. 23.—Vic. Branch, B.M.A.; ballot papers issued for election of office-bearers of Branch.
Nov. 24.—Vic. Branch, B.M.A., Council.
Nov. 25.—S. Aust. Branch, B.M.A..
Nov. 25.—Q. Branch, B.M.A., Council.
Nov. 26.—N.S.W. Branch, B.M.A..
Nov. 30.—Vic. Branch, B.M.A.; ballot papers returned to Victorian Branch for election of office-bearers of Branch.
Dec. 1.—Vic. Branch, B.M.A.; Annual General Meeting, election of office-bearers.
Dec. 7.—N.S.W. Branch, B.M.A., Ethics Committee.
Dec. 9.—Q. Branch, B.M.A., Council.
Dec. 10.—Q. Branch, B.M.A., Annual General Meeting.
Dec. 10.—N.S.W. Branch, B.M.A..
Dec. 10.—S. Aust. Branch, B.M.A., Council.

EDITORIAL NOTICES.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned.

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